Special Report Rapport spécial

Advisory: Bromethalin rodenticide — No known antidote

Robert Coppock

A member of the CVMA-SBCV Chapter recently brought to our attention a potential risk associated with bromethalin. The CVMA member expressed concern because there is no antidote for bromethalin. The CVMA decided to present a short description of the clinical toxicology of bromethalin.

Bromethalin is incorporated into poisonous baits. It is used because there is no known target species tolerance to bromethalin (1,2). Since there is no specific antidote for bromethalin intoxication, all that can be offered to veterinary and human patients is supportive care. In an attempt to reduce exposure and deaths in non-target species, Health Canada has placed restrictions on the methods of baiting in which bromethalin can be used (1,2).

Toxicology

Bromethalin is designed to be lethal as a one-time-ingestion rodenticide (3). This compound and its metabolite inhibit mitochondrial oxidative phosphorylation (3–9). The bromethalin parent compound is rapidly absorbed from the intestines and metabolized in the liver to the *N*-demethylated metabolite, which is a more potent inhibitor of mitochondrial respiration — ATP production. Bromethalin and its *N*-demethylated metabolite (desmethyl bromethalin) are lipid soluble and are distributed to the brain where they inhibit oxidative phosphorylation. Disruption of energy production causes microscopic cerebral edema and an increase in intracranial pressure. In the brain of birds, domestic animals, and humans, a cascade of edema and ion pump disruption-linked neurologic damage occurs resulting in a characteristic diffuse spongiosis of the white matter (4–9). The half-life in plasma is considered to be 5 to 6 days and hepatic-enteric recycling occurs.

Clinical presentation

Clinical presentation centers on CNS dysfunction with the onset of clinical signs occuring 4 hours to 7 days after ingestion (4–9). The bromethalin intoxication syndrome is dependent on the initial dose and has two forms. The first is hyperstimulation-like syndrome (convulsant syndrome) starting with tremors, hyperaesthesia, purposeless circling, unprovoked vocalization, hyperthermia, and seizures — opisthotonus progressing to depression, decerebrate posture, and death. The second form is the depressive — paralytic form and signs occur in 1 to 7 days and progressively increase over 1 to 2 weeks. Clinical signs generally begin with depression and disorientation and dysfunction of proprioception that advances to ataxia and rear limb weakness. As intoxication advances, partial paresis which progresses to generalized paralyses can be observed (4–9). Involuntary spinal reflex of the pelvic limb may be exaggerated and decreased response to nociceptive stimuli can occur. Bladder tone can be altered and dysuria can be observed.

Secondary poisoning

Secondary or relay poisoning could occur when dogs, cats, other animals, and birds consume animals that have ingested bromethalin (9,10).

Diagnosis

History of bromethalin ingestion is the best information leading to diagnosis (4–9). Astute owners may notice anomalous change in fecal color due to color markers in the ingested bait. Risk of being poisoned increases in animals that are off leash or allowed to roam. Bromethalin can be used in malicious poisonings. For animals presented without a history of ingestion, the clinical presentation places bromethalin in the intoxication portion of the differential diagnosis. Clinical pathological findings generally are within normal limits. Cerebrospinal fluid (CSF) pressure may or may not be increased, and when increased, the value is generally lower than increases caused by trauma. Other CSF parameters are generally within normal limits. Gross necropsy findings are nonspecific. Diagnosis is dependent on the findings from brain submitted for histopathology, and stomach-intestinal contents, fat, liver, kidney, and brain submitted for chemical analyses.

Treatment

Owners who observe animals ingesting bromethalin should immediately bring the animal to the clinic for emergency care (4–9). Thorough gastric lavage is the preferred treatment and induction of emesis is a second approach. Both treatments should be followed with activated charcoal. The first dose of activated charcoal may be given with an osmotic cathartic such as sorbitol. Activated charcoal should be repeated following the general dosing protocol (5). Electrolyte monitoring, especially sodium, is essential after activated charcoal and osmotic

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cathartics are given. Once clinical signs develop, treatments and supportive care are generally ineffective in animal and human patients (4–9).

References

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Reporting a Pesticide Incident

Health Canada advises that you do not have to be certain that a pesticide caused the effect in order to report it. You can report a pesticide incident directly to the pesticide company as listed on the product label. The company is required by law to report all incidents related to their products to Health Canada. You can also report the incident directly to Health Canada.

Instructions for Reporting a Domestic Animal Incident

http://www.hc-sc.gc.ca/cps-spc/pest/part/protect-proteger/incident/domest-animal-eng.php

Pesticide Incident Reporting Form — Domestic Animal Incident

http://www.hc-sc.gc.ca/cps-spc/alt_formats/pdf/pest/part/protect-proteger/incident/domest-animal-eng.pdf

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Book Review
Compte rendu de livre

Physiology of Domestic Animals. 2nd edition

Sjaastad OV, Sand O, Hove K. Oslo: Scandinavian Veterinary press. 2010. 804 pp. ISBN: 9788-2917-4397-3.

The target audience for this text is students of animal sciences and veterinary medicine. However, the simplicity of writing and basic conceptualization of key topics generally makes it more useful as a basic introductory text for either undergraduate students or high school students preparing for more intense college courses.

Contrary to the simplicity of the text, it is very well organized with topics building upon one another as you are guided through the chapters. Additionally, each chapter has questions included to help guide students to the key points that should be taken away from a chapter as well as a list of references for each chapter to guide further studies or more in depth information if needed.

Every chapter has a variety of charts and drawings, which are visually appealing and well described for the understanding of the reader. These images help to support the key ideas from each chapter as well as to engage visual learners in the study.

In general, this book would most benefit a veterinarian as a supplement for explaining information to clients in a more simplistic way that a layperson could understand. It could also be of benefit to a clinic as a quick reference for veterinary assistants desiring a better understanding of the importance of the work they are involved in.

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